14. (Amended) The expression vector of claim 2, wherein said secreted chemokine binds to a chemokine receptor.

15. (Amended) The expression vector of claim 14, wherein one or more amino acids are deleted from the N-terminus of the secreted chemokine.

C2

16. (Amended) The expression vector of claim 1, wherein said intracellular retention signal sequence directs a protein expressed from said single intrakine transcript to the endoplasmic reticulum, Golgi apparatus, a lysosome, an intracellular vesicle or other cellular compartment.

CONT.

17. (Amended) A method of inhibiting phenotypic expression of a chemokine receptor in a cell, wherein the method comprises blocking cell surface expression of said chemokine receptor by binding of said chemokine receptor with an intrakine.

23. (Amended) A method of inhibiting HIV infection of a cell, said method comprising phenotypically knocking out an HIV co-receptor in said cell by binding of said HIV co-receptor with an intrakine, wherein said phenotypic knock-out of said HIV co-receptor in said cell inhibits infection of said cell.

C3

24. (Amended) The method of claim 23, wherein said co-receptor is a C-C chemokine 5 receptor, a C-C chemokine 3 receptor, a C-C chemokine 1 receptor or a CXR4 receptor.

C4

33. (Amended) The method of claim 29, wherein said CC receptor is a C-C chemokine 5 receptor (CCR5), a C-C chemokine 3 receptor (CCR3), or a C-C chemokine 1 receptor (CCR1).

SUB D4

C5

35. (Amended) An expression vector for treatment of an HIV infection in a subject, wherein said expression vector includes:

an expression region which comprises:

a promoter;

an intracellular retention signal sequence encoding region; and

a chemokine encoding gene;

wherein said intracellular retention signal sequence and said chemokine encoding gene are expressed as a single intrakine transcript from said promoter; and

wherein when said expression vector is administered to lymphocytes, monocytes, macrophages or stem cells of said subject said cells exhibit a phenotypic knock out of an HIV co-receptor.

38. (Amended) A composition comprising the expression vector of claim 35 and a pharmaceutically acceptable solution.

39. (Amended) A method of increasing white blood cell count in a subject with an HIV infection comprising administering to said subject a pharmaceutical composition comprising lymphocytes, monocytes, macrophages or stem cells transduced with a vector of claim 1, thereby increasing white blood cell count in said subject with an HIV infection.

Please cancel claims 25-28 and 30-32, without prejudice to the inclusion of the subject matter contained therein in any later filed continuation or divisional application(s).

REMARKS

The present invention relates to novel methods and compositions for the treatment of HIV infection and for methods of conferring HIV resistance. The invention discloses, *inter alia*, methods of inhibiting HIV co-receptor cell surface expression using intracellular retained cytokines, *i.e.*, "intrakines," to bind to the receptors intracellularly and prevent transport of the receptors to the cell surface. The invention further relates to inhibiting HIV-1 infection using intrakines and secreted chemokines to competitively inhibit HIV-1 from binding with a co-receptor.

Claims 1-24, 29, and 33-39, are pending in the application. A copy of these claims as pending following the entry of the instant Amendment is enclosed herewith for the Examiner's convenience. Claims 25-28 and 30-32 have been cancelled herein without prejudice.